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EXAMINER	
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1634	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/655,915	ATTIE ET AL.	
	Examiner	Art Unit	
	Jehanne S. Sitton	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *24 January 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1 and 2* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1 and 2* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *1-2008*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. Currently, claims 1 and 2 are pending in the instant application. The following objections and rejections are either newly applied, as necessitated by amendment, or are reiterated. They constitute the complete set being presently applied to the instant Application. Response to Applicant's arguments follow. This action is FINAL.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

3. The New Matter Objection made at section 4 of the previous office action with regard to the amendment filed 8/15/2006 at Table 1, row 4 in the specification is withdrawn in view of the arguments and evidence provided in the Declaration under 37 CFR 1.132 by Dr. Susanne Clee, specifically at section 4a&d, 5, 6-9, 11 &12. The specification teaches that position 172 in mouse SORCS1 cDNA was mutated. Further the mouse sequence provided by Genbank Accession number AF195056 (which has not been updated and was publicly available at the time the application was filed and referenced in the specification) contains a 17 nt untranslated region as is evident by aligning the amino acid sequence for mouse SorCS1 taught in figure 2. Accordingly, when comparing this information, it is clear that the amino acid position in question in the mouse sequence should be amino acid 52, not 50, and that one of ordinary skill in the art would not only have found the error, but would have recognized the appropriate correction.

4. The rejection under 35 USC 112/second paragraph made in the previous office action is withdrawn in view of the amendments to the claims.

Maintained Objections

Specification

5. The amendment filed 5/4/2006 is objected to under 35 U.S.C. 132(a) because it introduces new matter into the disclosure. 35 U.S.C. 132(a) states that no amendment shall introduce new matter into the disclosure of the invention. The added material which is not supported by the original disclosure is as follows: In table 1, at page 4 of the specification, the amendment changes the amino acid position from 1140 to 1139 and 1150 to 1149. Additionally, the nucleotide sequences are changed from 3433 to 3436 and 3462 to 3465, respectively. However, the specification does not provide support for this specific change, nor does the specification provide any cDNA sequences for mouse isoforms SorCS1a, or c. At page 6, second paragraph, the response asserts that this change was made to correct a clerical error due to "inadvertent misnumbering". This argument has been thoroughly reviewed but was not found persuasive. As set forth in the MPEP 2163 (I) (B): "While there is no in haec verba requirement, newly added claim limitations must be supported in the specification through express, implicit, or inherent disclosure. An amendment to correct an obvious error does not constitute new matter where one skilled in the art would not only recognize the existence of the error in the specification, but also recognize the appropriate correction. In re Oda, 443 F.2d 1200, 170 USPQ 268 (CCPA 1971)." In the instant case, the specification does not provide any cDNA sequences for murine SorCS1 isoforms a or c. Although Figure 2 does provide amino acid sequences for murine Sorcs1a, b, and c isoforms, the 1139 to 1140 change is taught to occur in isoform a. Both positions 1139 and 1140 are taught to be Ser in the figure; however the figure provides absolutely no basis for changing the position from 1139 to 1140 and does not provide any

nucleotide sequence whatsoever. Turning to the change from 1149 to 1150, which is taught to occur in isoform c, it is noted that position 1150 shown in figure 2 is E, not T. Additionally, figure 2 specifically references position 1149, not 1150. Additionally, no nucleic acid sequence is provided by the specification. The changes to the nucleotide numbering at 3462 to 3465 is not supported by the specification. Accordingly, one skilled in the art, based on the guidance in the specification, would not have recognized the existence of the error or the appropriate correction.

Applicant is required to cancel the new matter in the reply to this Office Action.

Response to Arguments

6. The response asserts that the table was amended in the interest of sequence accuracy and completeness. This argument has been thoroughly reviewed but was not found persuasive as neither the declaration under 37 CFR 1.132 by Dr Clee nor the response provide any reasoning as to how one of skill in the art would be recognized the existence of this error or the appropriate correction. The objection is maintained.

New Grounds of Rejection necessitated by Amendment

Claim Rejections - 35 USC § 112

Written Description

7. Amended claims 1 and 2 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This includes a New Matter Rejection.

Amended claim 1 is drawn to a method of screening a human subject for susceptibility to type 2 diabetes by determining the SorCS1 cDNA sequence of that subject, deducing the amino acid sequence encoded thereby, and comparing it with SEQ ID NO: 4, wherein a difference of a threonine to isoleucine substitution at amino acid position 52 of the human SorCS1 amino acid sequence indicates that the subject is susceptible to developing type 2 diabetes. Amended claim 2, is drawn to a method of screening a human subject for susceptibility to type 2 diabetes by determining the SorCS1 cDNA sequence of the subject, comparing it with SEQ ID NO: 3, and screening for a difference of a cytosine to thymine substitution at nucleotide position 163 of the human SorCS1 cDNA sequence indicates that the subject is susceptible to developing type 2 diabetes.

The amended claims recite an amino acid change from threonine to isoleucine at position 52 or the a cytosine to thymine change at nucleic acid position 163 in human SorCS1, respectively, indicates that the human subject is a candidate for type 2 diabetes. However, the specification provides no basis for these specific nucleotide or amino acid positions in humans.

At para 00020 of the specification, the specification generally sets forth diagnostic use for examining humans for their SorCS1 gene and determining differences with respect to SEQ ID NO: 4. Although the specification recites specific positions in Table 1, these positions are with regard to differences found in B6 vs BTBR mice, both of which, were diabetic, albeit with differing severity. However, the specification does not appear to set forth screening methods for diabetes susceptibility in humans by determining any *particular* mutation or position. Accordingly, the newly added claims directed to screening methods in humans at a specific SorCS1 position appears to introduce new matter into the instantly claimed invention.

Additionally, as the mutations disclosed were found in mice not in humans, there is no evidence that such mutations would even exist in any human SorCS1 nucleic acid or protein sequence. The specific threonine to isoleucine change taught by the specification was in the mouse sequence. It is not known, nor does the specification teach if position 52 in humans is mutated, and if so, if it is mutated specifically to isoleucine. Further, even if amino acid position 52 were mutated in humans, there is no teaching that the mutation occurs specifically at position 163 of the human sequence. An amino acid position is determined by a 3 nucleotide codon in the nucleic acid coding sequence. A number of possible changes could occur in the nucleic acid sequence to result in an a change at a specific amino acid position. The specification provides absolutely no teaching or guidance that specifically, position 163 of the SorCS1 nucleic acid sequence in humans is mutated or that the mutation is a cytosine to thymine mutation. The declaration of Dr. Clee has been thoroughly reviewed. Although it provides analysis of the human SorCS1 amino acid and nucleic acid sequences relative to the mouse sequence with regard to the basis for amending the specification at table 1, row 4, the evidence provided by the declaration cannot supplement the disclosure in the specification. The teachings of the specification are limited and all of the disclosure with regard to specific SorCS1 nucleotide or amino acid variations in SorCS1 are with regard to variations found in mice not human sequences. The specification has no support for the specific amino acid or nucleotide mutations in human SorCS1 now recited in the amended claims.

Enablement

8. Amended claims 1-2 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. There are many factors to be considered when determining whether there is sufficient evidence to support determination that a disclosure does not satisfy the enablement requirements and whether any necessary experimentation is undue. These factors have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

Amended claim 1 is drawn to a method of screening a human subject for susceptibility to type 2 diabetes by determining the SorCS1 cDNA sequence of that subject, deducing the amino acid sequence encoded thereby, and comparing it with SEQ ID NO: 4, wherein a difference of a threonine to isoleucine substitution at amino acid position 52 of the human SorCS1 amino acid sequence indicates that the subject is susceptible to developing type 2 diabetes. Amended claim 2, is drawn to a method of screening a human subject for susceptibility to type 2 diabetes by determining the SorCS1 cDNA sequence of the subject, comparing it with SEQ ID NO: 3, and screening for a difference of a cytosine to thymine substitution at nucleotide position 163 of the

human SorCS1 cDNA sequence indicates that the subject is susceptible to developing type 2 diabetes.

Although the claims have been amended to recite methods of "screening", given the claim language in claim 1d and claim 2c, the nature of the invention requires the knowledge of predictive associations between position 52 relative to SEQ ID NO: 4 or position 163, relative to SEQ ID NO: 3 and susceptibility to developing type 2 diabetes in humans.

The claims recite "cDNA" of human SorCS1, however it is known that in mice, different isoforms of SorCS1 exist. The specification does not teach the different isoforms of human SorCS1.

The invention is in a class of inventions which the CAFC has characterized as 'the unpredictable arts such as chemistry and biology" (Mycolgen Plant Sci., Inc. v. Monsanto Co., 243 F.3d 1316, 1330 (Federal Circuit 2001)).

The specification teaches that the inventors began by narrowing the genetic region associated with severe type 2 diabetes to a 7 MB segment of mouse chromosome 19 (page 4, para 0017). The specification teaches that 2 genes previously found in the region were SorCS1 and SorCS3, which belong to a family sharing a large region of similarity including the VPS10 domain. The specification teaches that due to similarity with sortilin, SorCS1 and SorCS3 are expected to be involved in insulin-stimulated glucose transportation and in controlling body fat metabolism. The specification teaches that the 7MB region was characterized and that it was found that the only difference between severely diabetic mice and less severely affected mice was 3 mutations in SorCS1, leading to 3 amino acid changes (table 1). The specification, however, does not teach the specific function or activity of SorCS1. The specification does not

teach if other mutations occurred in other portions of the mouse genome that may be responsible for the severe form of diabetes observed in the mice.

The specification provides no teaching or working examples of any mutations in any portion of the SorCS1 gene in humans, or an association between SorCS1 alleles in a human subject and type II diabetes susceptibility. The specification asserts at page 3 that the SorCS1 gene in mice is "directly analogous" to the human gene, however this statement is unclear. The genes are not identical, and the meaning of "directly analogous" cannot be determined. For example, at table 1, the specification teaches different mutations at specific positions of mouse SorCS1. The specification teaches a mutation, at position 1140 from Ser to Phe, and at position 1150 from Ser to Pro. However, in humans position 1140 is Asp, and position 1150 (in SEQ ID NOS 4) is His. None of these amino acids are "directly analogous" to either amino acid found in mice at each position. Although, the specification has been amended to recite a mutation at position 52 from Thr to Ile (also found in SEQ ID NO: 4), the specification provides no teaching of the specific function or activity for SorCS1, or any of these 3 positions, accordingly the affect of each amino acid at such positions is unpredictable. Additionally, claims 1 and 3 broadly encompass any nucleotide change which leads to an isoleucine substitution at position 52, however only a single specific nucleotide substitution which leads to this alteration in mice is taught. The specification does not teach if the amino acid substitution is the causative mutation which leads to a more severe form of diabetes in mice, or if the nucleotide change may be in linkage with another allele. Therefore, given the lack of guidance from the specification as to any mutations in any region of the SorCS1 gene in humans, a teaching of the function of SorCS1 including critical amino acids and domains required for function, or a predictable correlation

between the presence of SorCS1 mutations and diabetes susceptibility in other species, the skilled artisan would be unable to predict an association between the claimed positions in the protein coding region or cDNA of the SorCS1 gene in humans and susceptibility to type 2 diabetes.

The specification's assertions with regard to putative SorCS1 activity is based on homology analysis with sortilin and the family of proteins that contains a VPS10 domain (page 4, end of para 00017). However, it is known for nucleic acids as well as proteins that even a single nucleotide or amino acid change or mutation can destroy or alter the function of a biomolecule in many instances, albeit not in all cases. The effect of these changes are largely unpredictable as to which ones have a significant effect verses not. The prior art does not teach the function of SorCS1 or how it is involved in type 2 diabetes. The post filing specifically date art provides some characterization of SorCS1 (see Hermey et al, JBC, vol. 278, Feb. 2003, pages 7390-7396), but teaches that neither the mature luminal domain nor any of the cytoplasmic domains of the different SorCS1 isoforms bound any of the ligands previously shown to interact with sortilin and SorLA, demonstrating sorCS1 is functionally different from the previously characterized Vps10-D family receptors (para bridging pages 7390-7391). Additionally, Hermey teaches that the different isoforms of SorCS1 have completely different cytoplasmic domains that mediate different trafficking in cells (abstract). It is clear that the art supports that SorCS1 has a different function than other Vps10 domain family members, and that the 3 different isoforms of SorCS1 do not function in the same manner where the different cytoplasmic domain for each isoform mediates different trafficking in cells.

Claims 1 and 2 appear to be drafted based on the amended specification's recitation that a difference was found in the SorCS1 gene between B6 and BTBR mice corresponding to amino acid

position 52. The specification asserts "It appears that the activity of the SorCS1 protein may determine islet mass. Alternatively, the SorCS1 protein may affect insulin secretion in pancreatic beta cells or insulin degradation in the kidney or liver" (page 8, para 00033), however the specification does not teach the function of SorCS1, or whether or how the change from Thr to Isoleucine altered the function or activity of the SorCS1 nucleic acid or protein such that the change provides an increased susceptibility to type 2 diabetes in mice. Accordingly, the affect of the mutation of Thr to Ile, is unpredictable in humans. The specification provides no guidance as to whether this mutation, or the other mutations listed in Table 1, occurs in a critical region or domain or how it affects the function or activity of a critical region or domain, such that the skilled artisan would be able to predict the same effects in humans. The specification provides no teaching or working examples that this mutation, or the other mutations listed in Table 1, exists in humans or would have a similar affect in humans. Kahn teaches that disruption of a specific gene in mouse models of diabetes does not necessarily provide a predictable correlation that any polymorphism in the corresponding human sequence would be similarly associated (Kahn, Cell, vol. 92, pages 593-596, 1998, cited in the IDS). Kahn teaches "Withers et al., reported that disruption of IRS-2 causes diabetes in mice. The most compelling aspect of this report is that inactivation of this single gene causes defects in both insulin action and insulin secretion." (page 593, last para of col. 2). However, Kahn further teaches "The parallels between the IRS-2 knockout mice and Type 2 diabetes in humans raises the tantalizing question as to whether human diabetes is caused by mutations in the IRS-2 gene. Disappointingly, studies in press in several populations, including Danish Caucasians… reveal no association between polymorphisms in the IRS-2 gene and Type 2 diabetes" (page 594, 2nd full para in col. 2).

The instant specification provides no teaching or guidance as to the role of critical amino acids in any of the isoforms of either murine or human SorCS1 nor how such are involved in susceptibility to type 2 diabetes. The specification provides no predictable association that any alteration, in any protein coding region or cDNA of the SorCS1 gene, including those claimed, in humans, let alone any species, is diagnostic or indicates a susceptibility for developing type 2 diabetes. No predictable correlation between the structural alterations in the mouse sequence and susceptibility for developing type 2 diabetes has been taught by the specification. The specification does not teach the function of SorCS1 nor how it's function, or lack of function, or altered function are predictably associated with type 2 diabetes.

The quantity of experimentation in this area is extremely large as it requires analysis of claimed positions in the SorCS1 gene to determine whether the isoleucine variant at position 52 of SEQ ID NO: 4, or the nucleotide variant T, at position 163 of SEQ ID NO: 3 is associated with type 2 diabetes. As neither the art nor the specification provide guidance as to whether the amino acids at such positions are critical to the function of SorCS1 or are in some way associated with diabetes susceptibility such analysis is replete with trial and error experimentation, with the outcome being unpredictable.

In order to practice the invention as claimed, one would first have to establish that a predictive relationship exists between the disclosed mutations and susceptibility to type 2 diabetes in humans. Such experimentation could involve functional analysis of a protein whose actual function has to be determined as well. The experimentation could also involve a large study of patients and controls to screen for mutations in SorCS1 in humans to determine whether the claimed mutations are associated with susceptibility to type 2 diabetes in humans. Such

analysis represents an inventive and unpredictable undertaking with each of the many intervening steps not providing any guarantee of success.

Thus, given the broad claims in an art whose nature is identified as unpredictable, the state of the prior art, the lack of guidance in the specification, the breadth of the claims and the quantity of experimentation necessary to practice the claimed invention, it would require undue experimentation to practice the invention commensurate in scope with the claims.

Response to Arguments

9. The response traverses the rejection. The response asserts that contrary to the remarks in the Office action, applicants have established that there is a predicable association between mouse and human SorCS1 gene and cites two references by the inventors; Clee et al; Endocrine Reviews, vol 28, pages 48-83, 2007 and Goodarzi et al; Diabetes, vol. 56, pages 1922-1929, 2007. This argument as well as the references have been thoroughly reviewed but were not found persuasive. First, it is noted that the arguments pertaining to the references have not been provided in declaration or affidavit format. The reason for requiring evidence in declaration or affidavit form is to obtain the assurances that any statements or representations made are correct as provided by 35 U.S.C. 25 and 18, U.S.C. 1001. In *Ex parte Gray* (10 USPQ2d 1923) the Courts held that conclusory statements made in publications could not substitute for a declaratory evidence filed under 37 CFR 1.132. Furthermore, in *Ex parte Ishizaka* (BdPatApp&Int 24 USPQ2d 1621), the Courts stated that 37 CFR 1.132 does not recognize the use of a publication as a substitute for a declaration. Consequently, a Declaration filed under 37 CFR 1.132 sworn by at least one of the instant inventors which cites/explains the relevant parts

of the references is needed. Additionally, the response does not set forth any particular teachings from the references cited to establish that the specification was enabling at the time of filing. As set forth in the MPEP 2164.05:

> "To overcome a prima facie case of lack of enablement, applicant must demonstrate by argument and/or evidence that the disclosure, as filed, would have enabled the claimed invention for one skilled in the art at the time of filing. This does not preclude applicant from providing a declaration after the filing date which demonstrates that the claimed invention works. However, the examiner should carefully compare the steps, materials, and conditions used in the experiments of the declaration with those disclosed in the application to make sure that they are commensurate in scope; i.e., that the experiments used the guidance in the specification as filed and what was well known to one of skill in the art. Such a showing also must be commensurate with the scope of the claimed invention, i.e., must bear a reasonable correlation to the scope of the claimed invention"

Notably, the specification and the claims sets forth that changes in the coding sequence of SorCS1 are associated with diabetes whereas Clee et al discusses SNPs in intronic sequences of SorCS1. However, a later dated publication cannot be used to supplement the disclosure in the specification. Further, Goodarzi et al does not provide any guidance that the claimed mutations even exist in humans, let alone are associated with type 2 diabetes susceptibility in humans. Accordingly, there is no nexus between the references cited and the instantly pending claims.

The response further asserts that the claims have been amended to recite screening methods rather than diagnostic methods. This argument has been thoroughly reviewed but was not found persuasive as the claims continue to recite a diagnostic relationship between the specific mutations and susceptibility to type 2 diabetes. The rejection is therefore maintained from the previous office action with regard to the amended claims.

Conclusion

10. No claims are allowed.

11. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

12. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-0752. The examiner can normally be reached Monday, Wednesday and Thursday from 9:00 AM to 3:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on (571) 272-0735. The fax phone number for this Group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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/Jehanne Sitton/
Primary Examiner
Art Unit 1634